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2444-3824/

Acute gastroenteritis and *Haemophilus parainfluenzae*: An unreported but predictable association☆

Gastroenteritis aguda y *Haemophilus parainfluenzae*: una asociación previsible pero no reportada

The term acute gastroenteritis is applied to symptoms of diarrhoea or vomiting ascribable to an infection of the proximal segment of the small intestine or the colon. It is amongst some of the most common infectious diseases and it involves a heightened morbimortality, particularly among the elderly, malnourished and those with underlying illnesses.[1]

The *Haemophilus* spp. coccobacilli make up the normal bacterial flora of the upper respiratory tract, where *Haemophilus influenzae* is the predominant type. In turn, *Haemophilus parainfluenzae (H. parainfluenzae)* has been related to different respiratory tract infections and conjunctivitis. Less commonly, it can cause infective endocarditis and more rarely white tissue abscesses, septic arthritis, genital tract infections, osteomyelitis, wound infections and in very rare cases, meningitis and brain abscesses.[2,3] In recent years, cases of intrabdominal infections of the bile duct, hepatic or pancreatic abscesses, peritonitis and appendicitis have been published.[3–7]

In this article we propose to describe a case of bacterial gastroenteritis by *H. parainfluenzae*.

A 43-year-old male born in Spain, with no previous pathologies of interest nor recent trips, that was seen at our centre presenting a 4-day history consisting of colic-like abdominal pain, vomiting and diarrhoea with no pathological products, accompanied by a high fever and chills.

Upon physical examination, he displayed a heart rate of 103 bpm, blood pressure of 98/63 mmHg and a temperature of 38 °C, with no signs of dehydration. Upon abdominal examination, diffuse pain was noted, more intense in the epigastrium but with no signs of peritonitis and no organomegalies were felt. He also presented cold-sores. The rest of the physical examination was normal.

The blood test showed: white blood cells 12,100 μl with a neutrophil percentage of 82.5%, platelets 95,000 μl, haemoglobin 12.1 mg/dl, C-reactive protein 163.5 mg/l with normal kidney liver function, water-electrolyte balance, amylase and coagulation. HIV infection was ruled out. Cultures were started he was admitted to hospital, receiving Ciprofloxacin as treatment. Whilst he was admitted, he underwent an abdominal CAT scan that reported the presence of findings suggestive of infectious or inflammatory non-specific colitis, affecting segments of the ascending colon. The rest of his abdominal structures were normal. On his fourth day in hospital, *H. parainfluenzae* was isolated in the 2 blood cultures, sensitive to most antibiotics, including quinolones. The stool culture was negative, showing only the presence of conventional mixed flora.

His clinical progress was favourable. In the subsequent outpatient follow-ups 2 months later, the patient remained asymptomatic.

We believe that the patient showed symptoms of acute gastroenteritis caused by *H. parainfluenzae*. The initial clinical symptoms, radiological findings and absence of data suggestive of an alternative diagnosis paired with the isolation of the pathogen in the blood enabled this suspected diagnosis to be confirmed. We cannot rule out that the patient may suffer gastroenteritis from another microorganism, which would allow *H. parainfluenzae* to pass through to the bloodstream. However, taking into account the symptoms of bacteraemia and the absence of other intestinal pathogens, this hypothesised diagnosis does not seem to be substantiated. We must point out that the current culture mediums are not designed to isolate *H. parainfluenzae* in the faeces samples obtained for stool cultures.

In recent years, studies of the bacterial flora in the GI tract have been gaining great importance for their potential relationship with different diseases such as: pseudomembranous colitis, inflammatory bowel disease, irritable bowel syndrome and even chronic constipation and obesity. In this regard, potentially pathogenic microorganisms that were formerly considered to be completely unrelated to this field have gained greater relevance, like with *H. parainfluenzae*, assiduously isolated between the microbacterial flora of the GI tract.[8]

Thus, Palmer GG isolated *H. parainfluenzae* in the intestinal mucosa and suggested that it could possibly act as a pathogen if the gastrointestinal acidity decreased or if the mucosa was altered.[9] Later, Mégraud et al., postulated that the GI tract might work as a reservoir for *H. parainfluenzae*, and they also suggested a possible relationship between the cases of bacteraemia caused by this microorganism, with apparently unknown origins and intercurrent gastrointestinal processes.[10]

Ultimately, we consider out patient to represent a first documented case of acute gastroenteritis by

☆ Please cite this article as: Olivart M, Galera E, Falguera M. Gastroenteritis aguda y *Haemophilus parainfluenzae*: una asociación previsible pero no reportada. Gastroenterol Hepatol. 2017;40:23–24.

H. parainfluenzae, a finding that should not be surprising based on recent research on the gastrointestinal habitat.

Funding

No funding was received to draft our manuscript.

Conflicts of interest

The authors declare that there is no conflict of interest with regard to the article.

References

1. Steiner T, Guerrant R. Principios y síndromes de infección entérica. In: Mandell G, Bennet J, Dolin R, editors. Enfermedades infecciosas. 7the ed. Barcelona: Elsevier; 2012. p. 1343–56.
2. Murphy T. Género *Haemophilus* (incluidos *H. influenzae* y chancroide). In: Mandell G, Bennet J, Dolin R, editors. Enfermedades infecciosas. 7th ed. Barcelona: Elseiver; 2012. p. 2915–23.
3. Frankard J, Rodríguez-Villalobos H, Struelens MJ, Jacobs F. *Haemophilus parainfluenzae*: an undiagnosed pathogen of biliary tract infections. Eur J Clin Microbiol Infect Dis. 2004;23:46–8.
4. Bottone EJ, Zhang DY. *Haemophilus parainfluenzae* biliary tract infection: rationale for an ascending route of infection from the gastrointestinal tract. J Clin Microbiol. 1995;33:3042–3.
5. Friedl J, Stift A, Berlakovich GA, Taucher S, Gnant M, Seteinenger R, et al. *Haemophilus parainfluenzae*. Liver abscess after successful liver transplantation. J Clin Microbiol. 1998;36:818–9.
6. Martín-Berra M, Navarro-López V, Sirvent E, Montiel J. Infección de pseudoquiste pancreático por *Haemophilus parainfluenzae*. Rev Med Chile. 2011;139:215–7.
7. Betriu C, Coronel F, Martín P, Picazo JJ. Peritonitis caused by *Haemophilus parainfluenzae* in a patient undergoing continuous ambulatory peritoneal dialysis. J Clin Microbiol. 1999;39:3074–5.
8. Aroniadis O, Brandt L. Fecal microbiota transplantation: past, present and future. Curr Opin Gatroenterol. 2013;29:79–84.
9. Palmer GG. Haemophili in faeces. J Med Microbiol. 1981;14:147–80.
10. Mégraud F, Bébéar C, Dabernat H, Delmas C. *Haemophilus* species in the human gastrointestinal tract. Eur J Clin Microbiol Infect Dis. 1988;7:437–8.

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2444-3824/

Our experience in the surgical treatment of liver PEComa☆

Nuestra experiencia en el tratamiento del PEComa hepático

Liver PEComas are extremely rare and differential diagnosis is complex. The treatment of choice is surgical removal. We present our experience (4 cases in 6 years) and a literature review.

Case 1

A 46-year-old female with a history of right hepatectomy in 1995 due to a hepatocellular carcinoma (HCC).

After 10 years, damage to segment II was found. The biopsy showed non-tumour hepatocytes. Four years later, the magnetic resonance imaging (MRI) scan showed a growth in size, hypervascular behaviour and a biopsy consistent with HCC.

There was 2.4 cm of damage in segment II on the intraoperative ultrasound, and a limited resection was performed.

The final outcome was PEComa.

The histological preparations from the prior resection were requested and they were confirmed as a match with a liver PEComa (company unknown in 1995).

Case 2

A 45-year-old female with a malignant melanoma previously resected in her lower left limb, Clark IV. After 8 years, the follow-up CT scan found a solid 25 mm lesion on the MRI described as: hypervascular, hyperintense in T1 and hypointense in T2.

Biopsy consistent with PEComa.

On a morphologically normal liver, the intraoperative ultrasound identified a 2.5 cm hyperechoic lesion, which was well defined in segment VIII. A limited resection was performed.

Case 3

A 48-year-old female diagnosed with ocular choroidal melanoma. The extension study identified a 7 cm mass in the liver hilum with a liver PEComa biopsy.

☆ Please cite this article as: Otegi Altolagirre I, de Miguel Valencia M, Sánchez Acedo P, Zazpe Ripa C, Tarifa Castilla A, Herrera Cabezón J, et al. Nuestra experiencia en el tratamiento del PEComa hepático. Gastroenterol Hepatol. 2017;40:24–28.